In the Claims:

1. (Currently amended) A method for ~~predicting~~ determining the relative affinity for binding to a ligand of a peptide that competes with a polypeptide for binding to the ligand, which peptide is ~~derived from~~ produced by a phagemid clone present in a phage-displayed library, which method comprises incubating a phagemid clone ~~corresponding to~~ displaying the peptide with the polypeptide in the presence of the ligand, serially diluting the ~~phage~~ phagemid clone, and measuring the degree to which binding of the phagemid clone to the ligand is inhibited by the peptide displayed on said phagemid clone, wherein the phagemid clone that is inhibited only at low ~~phage~~ phagemid concentrations has a higher affinity for the ligand than a phagemid clone that is inhibited at both high and low ~~phage~~ phagemid concentrations, wherein the degree to which binding of the phagemid clone to the ligand is inhibited by the peptide determines the relative affinity for binding of the peptide to the ligand.

2. (Earlier presented) The method of claim 1 wherein the ligand is an IGF binding protein and the polypeptide is an IGF.

In the Specification:

The paragraph, beginning at page 132, line 33, has been amended as follows:

After incubation of normal human serum with ^{125}I-IGF-I for 16 hr, three peaks of radioactivity were observed following FPLC-SEC (Fig. ~~42~~ 41). These peaks likely correspond to the following ^{125}I-IGF-I complexes: ~150 kDa (^{125}I-IGF-I:IGFBP-3:ALS); ~44 kDa (^{125}I-IGF-I:IGFBPs 1-4); ~7 kDa (free ^{125}I-IGF-I). As can be seen in Figure ~~42~~ 41, addition of the IGF mutant to the serum resulted in a time-dependent decrease in ^{125}I-IGF-I associated with the ^{125}I-IGF-I:IGFBP complexes and an increase in the amount of free ^{125}I-IGF-I. These results are outlined in the above table. The ^{125}-I-IGF-I was more readily displaced from the ~44 kDa than the ~150 kDa ^{125}I-IGF-I:IGFBP complex, suggesting that IGF-I bound to the lower molecular weight IGFBPs is in a more bioavailable form. These data clearly indicate that the IGF mutant has the ability to displace IGF-I from endogenous IGFBPs present in normal human serum and therefore is likely to be active *in vivo* in humans.

The paragraph, beginning at page 134, line 17, has been amended as follows: